tionship. He was discharged with follow-up by a psychiatrist who works extensively with young adults.

The Internet has become a ubiquitous source of information for both professionals and patients alike. A large number of web sites and discussion groups provide information identifying methods of self-harm. Of the methods described in this case, I was able to locate the latter easily. What is less clear is information detailing how frequently patients use these resources and potential features that would allow at-risk individuals to be identified. Since the methods described on the Internet are potentially more lethal, being able to identify this group accurately is crucial in risk assessment. The number of reported cases is small. In three cases (reference 2 and this letter), patients were diagnosed with personality disorders or borderline mental retardation, indicating that they likely would use rigid, concrete, or maladaptive coping strategies. Creating a profile associated with individuals using Internet-derived suicide plans will require more study. In the meantime, clinicians should be alert to the dangers of Internet use by their suicidal patients.

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Clozapine-Induced Acute Interstitial Nephritis

TO THE EDITOR: A patient in our institution developed acute renal failure that was highly suggestive of acute interstitial nephritis after induction of the antipsychotic clozapine. There are few published reports documenting this side effect, the majority occurring in Great Britain, and none has appeared in the psychiatric literature (1). Therefore, we report a case of clozapine-induced acute interstitial nephritis in an effort to prevent further episodes of delayed diagnosis and treatment of this rare complication.

Mr. A was a healthy 33-year-old man with paranoid schizophrenia who was transferred to our institution after the onset of recurrent hallucinations. He was stabilized at another institution with 1500 mg of valproic acid at bedtime, 80 mg b.i.d. of ziprasidone, and 800 mg t.i.d. of gabapentin. His medications were subsequently changed to 500 mg b.i.d. of valproic acid, 800 mg at bedtime of gabapentin and 400 mg in the morning, and 2 mg of risperidone at bedtime. Minimal improvement was noted, and on the third week, 25 mg of clozapine at bedtime was started and titrated to 100 mg. At the initiation of treatment, all of Mr. A's laboratory blood work had been normal. One week later, his eosinophil count was slightly elevated at 6.1% (absolute count=0.49 10×9/liter). On the second week after the introduction of clozapine, he developed nausea, vomiting, a sore throat, diarrhea, a fever of 104°F, and a positive urinalysis (leukocyte esterase, WBC casts, 2+ protein, 1+ hemoglobin, 10–50 WBCs). Trimethoprim-sulfamethoxazole was started to treat a suspected urinary tract infection, and laboratory samples drawn at that time showed a sodium level of 132 mEq/liter, a BUN level of 87 mg/dl, and a creatinine level of 9.7 mg/dl. Mr. A was diagnosed with acute renal failure and transferred to a tertiary care center.

At that point, all medications were stopped except for valproic acid, and Mr. A was given intravenous hydration. An ultrasound of his kidneys showed bilateral echogenic kidneys without hydronephrosis, moderate renal disease, and glomerular nephritis. Mr. A maintained good urine output throughout his hospital stay. Over 1 week, he was monitored, and his WBC rose from 12.5 to 22.3 (10×9/liter), his eosinophil count rose to 11% (absolute count=2.5 10×9/liter), but his BUN and creatinine levels decreased to 76 mg/dl and 4.1 mg/dl, respectively, signaling at least partial return of renal function. He was discharged after a short hospital stay without further significant treatment.

Most often, acute interstitial nephritis is drug induced and appears soon after initiation of new drugs with the clinical triad of rash, fever, and eosinophilia. While multiple drugs have been implicated as possible etiologies of acute interstitial nephritis, the patient's previous tolerance of all other medications and the chronological onset of his symptoms after the initiation of clozapine suggest that this case most likely represents clozapine-induced acute interstitial nephritis. As weekly blood draws to detect agranulocytosis are the standard of care for the first 6 months of treatment with clozapine, we suggest that a rising eosinophil count in the setting of recent initiation of clozapine therapy may be a predictor of drug-induced acute interstitial nephritis and should be managed as such.

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Elevated Binding of D8/17-Specific Monoclonal Antibody to B Lymphocytes in Tic Disorder Patients

TO THE EDITOR: Several groups have independently reported elevated D8/17 expression on B lymphocytes in patients with a tic or related disorders, making this a promising peripheral blood marker for these conditions (1). However, concerns regarding insufficient sensitivity of the D8/17 assay have recently been raised (2). This prompted us to reanalyze the data of our published study on D8/17 B cell expression in tic disorder patients compared with healthy subjects (1). To our knowledge, our study was the only one reported thus far that used both flow cytometry and a control immunoglobulin M (IgM) monoclonal antibody. For this purpose, we used MOC32, an IgM monoclonal antibody that is directed against a neuroendocrine antigen of epithelial origin of small cell lung cancer cells. In contrast to previous studies, we did not assess a percentage of D8/17-positive B cells, since our flow cytometric analysis did not indicate a separate subpopulation of D8/17 positive B cells. Instead, we calculated D8/17 B cell overexpression by subtracting the mean fluorescence inten-